19 **Hui SL**, Slemenda CW, Johnston CC Jr. Baseline measurement of bone mass predicts fracture in white women. *Ann Intern Med* 1989;**111**:355–61.
20 **Matkovic V**, Kostial K, Simonovic I, *et al*. Bone status and fracture rates in two regions of Yugoslavia. *Am J Clin Nutr* 1979;**32**:540–9.
21 **Lloyd T**, Myers C, Buchanan JR, *et al*. Collegiate women athletes with irregular menses during adolescence have decreased bone density. *Obstet Gynecol* 1988;**72**:639–42.
22 **Bennell K**, Matheson G, Meeuwisse W, *et al*. Risk factors for stress fractures. *Sports Med* 1999;**28**:91–122.
23 **Bennell KL**, Malcolm SA, Thomas SA, *et al*. Risk factors for stress fractures in track and field athletes. A twelve-month prospective study. *Am J Sports Med* 1996;**24**:810–18.
24 **Lloyd T**, Triantafyllou SJ, Baker ER, *et al*. Women athletes with menstrual irregularity have increased musculoskeletal injuries. *Med Sci Sports Exerc* 1986;**18**:374–9.
25 **Lindberg JS**, Fears WB, Hunt MM, *et al*. Exercise-induced amenorrhea and bone density. *Ann Intern Med* 1984;**101**:647–8.
26 **Marcus R**, Cann C, Madvig P, *et al*. Menstrual function and bone mass in elite women distance runners. Endocrine and metabolic features. *Ann Intern Med* 1985;**102**:158–63.
27 **Myburgh KH**, Hutchins J, Fataar AB, *et al*. Low bone density is an etiologic factor for stress fractures in athletes. *Ann Intern Med* 1990;**113**:754–9.
28 **Nattiv A**. Stress fractures and bone health in track and field athletes. *J Sci Med Sport* 2000;**3**:268–79.
29 **Warren MP**, Brooks-Gunn J, Hamilton LH, *et al*. Scoliosis and fractures in young ballet dancers. Relation to delayed menarche and secondary amenorrhea. *N Engl J Med* 1986;**314**:1348–53.
30 **Loucks AB**, Heath EM. Induction of low-T_3 syndrome in exercising women occurs at a threshold of energy availability. *Am J Physiol* 1994;**266**:R817–23.
31 **Ihle R**, Loucks AB. Dose-response relationships between energy availability and bone turnover in young exercising women. *J Bone Miner Res* 2004;**19**:1231–40.
32 **Bullen BA**, Skrinar GS, Beitins IZ, *et al*. Induction of menstrual disorders by strenuous exercise in untrained women. *N Engl J Med* 1985;**312**:1349–53.
33 **Leibel RL**, Rosenbaum M, Hirsch J. Changes in energy expenditure resulting from altered body weight. *N Engl J Med* 1995;**332**:621–8.
34 **Deuster PA**, Kyle SB, Moser PB, *et al*. Nutritional intakes and status of highly trained amenorrheic and eumenorrheic women runners. *Fertil Steril* 1986;**46**:636–43.
35 **Drinkwater BL**, Nilson K, Chesnut CH III, *et al*. Bone mineral content of amenorrheic and eumenorrheic athletes. *N Engl J Med* 1984;**311**:277–81.
36 **Kaiserauer S**, Snyder AC, Sleeper M, *et al*. Nutritional, physiological, and menstrual status of distance runners. *Med Sci Sports Exerc* 1989;**21**:120–5.
37 **Myerson M**, Gutin B, Warren MP, *et al*. Resting metabolic rate and energy balance in amenorrheic and eumenorrheic runners. *Med Sci Sports Exerc* 1991;**23**:15–22.
38 **Nelson ME**, Fisher EC, Catsos PD, *et al*. Diet and bone status in amenorrheic runners. *Am J Clin Nutr* 1986;**43**:910–16.
39 **Wilmore JH**, Wambsgans KC, Brenner M, *et al*. Is there energy conservation in amenorrheic compared with eumenorrheic distance runners? *J Appl Physiol* 1992;**72**:15–22.
40 **Filicori M**, Flamigni C, Campaniello E, *et al*. Evidence for a specific role of GnRH pulse frequency in the control of the human menstrual cycle. *Am J Physiol* 1989;**257**:E930–6.
41 **Loucks AB**, Mortola JF, Girton L, *et al*. Alterations in the hypothalamic-pituitary-ovarian and the hypothalamic-pituitary-adrenal axes in athletic women. *J Clin Endocrinol Metab* 1989;**68**:402–11.
42 **Loucks AB**, Verdun M, Heath EM. Low energy availability, not stress of exercise, alters LH pulsatility in exercising women. *J Appl Physiol* 1998;**84**:37–46.
43 **Williams NI**, Caston-Balderrama AL, Helmreich DL, *et al*. Longitudinal changes in reproductive hormones and menstrual cyclicity in cynomolgus monkeys during strenuous exercise training: abrupt transition to exercise-induced amenorrhea. *Endocrinology* 2001;**142**:2381–9.
44 **Williams NI**, Helmreich DL, Parfitt DB, *et al*. Evidence for a causal role of low energy availability in the induction of menstrual cycle disturbances during strenuous exercise training. *J Clin Endocrinol Metab* 2001;**86**:5184–93.
45 **James AP**, Lorraine M, Cullen D, *et al*. Muscle glycogen supercompensation: absence of a gender-related difference. *Eur J Appl Physiol* 2001;**85**:533–8.
46 **Tarnopolsky MA**, Zawada C, Richmond LB, *et al*. Gender differences in carbohydrate loading are related to energy intake. *J Appl Physiol* 2001;**91**:225–30.
47 **Burke LM**, Cox GR, Culmmings NK, *et al*. Guidelines for daily carbohydrate intake: do athletes achieve them? *Sports Med* 2001;**31**:267–99.
48 **Loucks AB**, Callister R. Induction and prevention of low-T_3 syndrome in exercising women. *Am J Physiol* 1993;**264**:R924–30.
49 **Loucks AB**, Verdun M. Slow restoration of LH pulsatility by refeeding in energetically disrupted women. *Am J Physiol* 1998;**275**:R1218–26.
50 **De Souza MJ**, Miller BE, Loucks AB, *et al*. High frequency of luteal phase deficiency and anovulation in recreational women runners: blunted elevation in follicle-stimulating hormone observed during luteal-follicular transition. *J Clin Endocrinol Metab* 1998;**83**:4220–32.
51 **Kopp-Woodroffe SA**, Manore MM, Dueck CA, *et al*. Energy and nutrient status of amenorrheic athletes participating in a diet and exercise training intervention program. *Int J Sport Nutr* 1999;**9**:70–88.
52 **Torstveit MK**, Sundgot-Borgen J. The female athlete triad: are elite athletes at increased risk? *Med Sci Sports Exerc* 2005;**37**:184–93.
53 **Loucks AB**. Methodological problems in studying the female athlete triad. *Med Sci Sports Exerc* 2006;**38**:1020.
54 **Williams NI**, De Souza MJ. Female athlete triad errors and misunderstandings. *Med Sci Sports Exerc* 2006;**38**:1021.

Refutation of the myth of the female athlete triad

We are pleased to respond to *Refutation of ''the myth of the female athlete triad''* by Loucks; however, to respond in a point-by-point manner to each and every issue of contention would prove quite tiresome, and, more than likely, futile. Therefore, our response will focus on the more general issues of science and language, with particular attention to the translation (ie, application) of laboratory findings into practice and then into policy intended to affect collective behaviours. In doing so, we wish to remind the reader that there are guidelines governing the delicate balance between science and practice. We have described these guidelines previously with regard to the triad,[1] and wish to reiterate that they were developed to prevent practitioners, policy makers and regulators from reacting either too hastily to incomplete science or too slowly to sound science. We continue to maintain that the science pertaining to the female athlete triad is less than complete. Therefore, any attempt to influence practice or policy with regard to the triad should be made with extreme caution, as these efforts may be misguided at this time.

Loucks *et al*[2–5] have made a landmark contribution to women's health by identifying a mechanism (low energy availability, independent of exercise stress) by which exercise disrupts leutinising hormone pulsatility. That this mechanism was identified using the strictest of experimental methods lends substantial internal validity to their findings. Moreover, menstrual function changes were then reversed when energy availability was returned to match energy expenditure, thereby giving even more credibility to the data (as well as to our argument). Whether one feels comfortable in generalising these short-term laboratory-based data from non-athletes and primates to the general population of female athletes who train and compete over many years under real-life conditions is another matter altogether. Dr Loucks *et al*[6 7] may have little use for observational studies; however, Torstveist and Sundgot-Borgen have contributed the best population-based data to date on the issue of the triad.[8 9] At the very least, they provide prevalence estimates of the individual triad components and in toto that were collected from real athletes and a representative control group, rather than from small, select laboratory samples of non-athletes. But here lies the scientific quandary: the experimental data provide us with a necessary biological mechanism, yet the epidemiological data provide little evidence of the pathophysiological relevance of this mechanism to health and function among the population. Which of these two components of science is more important to practice and policy?

In any case, as important as the identification of a biologically plausible mechanism is to the aetiological relationship between exercise and menstrual function changes, one proposed mechanism alone is hardly sufficient to predict the purported risk of triad-related pathophysiology over the lifespan among women. In fact, a constellation of host and environmental factors will also influence one's susceptibility or resistance to menstrual function changes and bone loss, probably even in the presence of low energy availability. If this were not the case, most currently competitive athletes would be sitting on the sidelines with stress fractures, and the infertility clinics currently would be overflowing with former athletes. Needless to say, neither of these scenarios is occurring. Presumably, we will need to wait 30–40 more years to see if these same former athletes are filling the nursing homes with hip fractures because of low bone accrual during adolescence. This would be the value of large-scale epidemiological research that followed up a cohort of female athletes (and controls) through their competitive years and beyond into middle and older age to study longitudinally the influence of low energy availability in adolescence and young adulthood on infertility and osteoporosis later on. At the very least, former National Collegiate Athletic Association (NCAA) athletes (those first awardees of athletic scholarships from 1975 to 1980) currently can be assessed cross-sectionally in middle age and compared with their non-athletic peers. Oddly, we have not encountered any such data, presumably because it is expensive to perform these studies properly with objective biomarkers and measures of energy availability, endocrine function and bone resorption. On the other hand, if the triad is really an issue of such high clinical and public health significance as implied, one would think that the National Institutes of Health, the Centers for Disease Control and Prevention, the World Health Organization or at least the NCAA itself would be willing to fund such a venture.

We did not contribute to the writing of the female athlete triad position stand, or to the

papers that followed on the same topic, and therefore we comment only on what we read. Language is extremely important in communicating scientific findings to peers and, more importantly, to the public. Thus, it is important to be as precise as possible. The public, in turn, must weigh a given risk against other risks they are willing to assume on a daily basis (eg, driving a car, smoking cigarettes, eating French fries or not exercising), and this ''risk mix'' ultimately will influence individual risk perception and behaviour. It is our opinion that in many instances concerning the female athlete triad, the data do not match the sensational language often used to warn young girls and women of ''the risks associated with exercise''. Further, although the social marketing value of the catchphrase ''female athlete triad'' is high, it connotes something bigger than what can actually be measured properly, and, frankly, is insulting to most women athletes who train and compete hard, bear children, and continue towards a healthy and successful older age. Indeed, if undernutrition (ie, low energy availability) in sports is the primary issue at hand, then any position stand and subsequent papers to this effect should be titled as such, and should be directed towards health consequences for male and female athletes. Finally, a position stand from the American College of Sports Medicine or any other organisation attempting to influence practice and policy should be evidence based and should rely on the highest quality data and not primarily on those generated from consensus or from the same group of researchers.

We remain grateful for the opportunities to state our opposing views on the female athlete triad. Such opportunities have allowed us to confront several difficult issues that are sociopolitical as well as scientific. As scientists, we should (with respect) agree to disagree on the specific areas of contention concerning the triad and trust that individuals will make informed choices about their own behaviour based on the best available knowledge.

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References

1 **DiPietro L**, Stachenfeld NS. The female athlete triad myth. *Med Sci Sports Exerc* 2006;**38**:795.
2 **Loucks AB**, Thuma JR. Leutinizing hormone pulsatility is disrupted at a threshold of energy availability in regularly menstruating women. *J Clin Endocrinol Metab* 2003;**88**:297–311.
3 **Loucks AB**, Heath EM. Induction of low-T_3 syndrome in exercising women occurs at a threshold of energy availability. *Am J Physiol* 1994;**266**:R817–23.
4 **Loucks AB**, Verdun M, Heath EM. Low energy availability, not the stress of exercise, alters LH pulsatility in exercising women. *J Appl Physiol* 1998;**84**:37–46.
5 **Williams NI**, Caston-Balderrama AL, Helmreich DL, *et al*. Longitudinal changes in reproductive hormones and menstrual cyclicity in cynomolgus monkeys during strenuous exercise training: abrupt transition to exercise-induced amenorrhea. *Endocrinology* 2001;**142**:2381–9.
6 **Loucks AB**. Methodological problems in studying the female athlete triad. *Med Sci Sports Exerc* 2006;**38**:1020.
7 **Williams NI**, De Souza MJ. Female athlete triad errors and misunderstandings. *Med Sci Sports Exerc* 2006;**38**:1021.
8 **Torstveit MK**, Sundgot-Borgen J. The female athlete triad: are elite athletes at increased risk? *Med Sci Sports Exerc* 2005;**37**:184–93.
9 **Torstveit MK**, Sundgot-Borgen J. The female athlete triad exists in both elite athletes and controls. *Med Sci Sports Exerc* 2005;**37**:1449–59.

Correction of misinterpretations and misrepresentations of the female athlete triad

The recent commentary by DiPietro and Stachenfeld[1] is of great concern because it lacks scientific accuracy in the interpretation of data regarding the female athlete triad and promotes an unfounded fear that triad-related data will discourage girls and women from participating in sports.

DiPietro and Stachenfeld state that they wish to ''share comments and opinions which challenge many of the causal assumptions proposed in the current literature on this topic''. They first attack the triad from a sociological perspective, warning readers that decades of progress of women in sports is at risk owing to ''the creation of yet another form of female specific pathology''. They are concerned that ''Triad related data may be misinterpreted and used as justification for setting health and social policies that may ultimately counter the US Public Health Service efforts to promote the benefits of athletic participation and an active lifestyle among children and adolescents''. Using this logic, researchers and health professionals should abandon their efforts to understand and prevent anterior cruciate ligament injuries in women, which occur at a rate of 6–8 times that in men,[2] for fear that attention to this ''female specific pathology'' would discourage participation in sports. Interestingly, although over one hundred studies document the existence of menstrual disturbances, disordered eating and low bone mass in exercising women,[3 4] DiPietro and Stachenfeld do not offer a single data point in support of their ''opinion'' that education about the triad might discourage the participation of girls and women in physical activity. In fact, actual data suggest the opposite. The National Federation of State High Schools Associations (NFHS) *High School Athletics Participation Survey*[5] reports that the number of girls participating in high school sports set an all-time record in 2004–5, soaring to 2 908 390 participants and representing a 13% increase compared with the 1997–8 rates. Data from the US National Collegiate Athletic Association (NCAA) *Sports Sponsorship and Participation Rates Report*[6] also present similarly increasing numbers of women participating in NCAA sports at the collegiate level. Comparisons of 1997–8 participation rates with the 2004–5 rates show a 51% increase in women's participation at the collegiate level.[6] Thus, participation rates of girls and women at the high school and collegiate levels have continued to rise since the 1997 publication of the triad, and these numbers exceed the increases in participation rates observed in boys and men's sports at both the high school (9%) and collegiate (9%) levels.[5 6] Consequently, the Fédération Internationale de Football Association (FIFA) estimates that worldwide, by 2010 more women than men will be playing football.[7]

Therefore, we challenge DiPietro and Stachenfeld to support their ''opinion'' by designing, executing and publishing in a peer reviewed journal a prospective randomised trial to directly test their hypothesis that educating the public about the possible unhealthy effects of the triad discourages participation in sports. They might also assess how many wrestlers discontinued their sport when, upon the publication of an American College of Sports Medicine (ACSM) position stand on unhealthy weight loss practices in wrestling,[8] new rules regarding body weight management in collegiate wrestling were implemented by the NCAA. They might also assess how many people did not take up or abandoned an exercise routine after another ACSM position stand[9] warned of the dangers of dehydration and running in the heat. Meanwhile, as we wait for these important datasets, it is likely that the benefits of education and policy about the health hazards of the triad will increase the numbers of girls and women who can maintain a healthy and active lifestyle over their lifespan because they were warned about the triad in time to prevent injuries, decrements in performance and loss of bone mass—just as objective evidence has shown that the NCAA weight management programme has been effective in reducing the practice of unhealthy weight loss behaviours among wrestlers.[10]

DiPietro and Stachenfeld define the triad as ''a syndrome consisting of three necessary components: (a) disordered eating; (b) amenorrhea; (c) osteoporosis''. This statement is factually incorrect. In the ACSM 1997 position stand,[11] the triad is defined as the ''existence of one or more components of the Female Athlete triad, alone or combination, that pose significant health risks to physically active athletic girls and women''. Thus, the simultaneous existence of all three components is not necessary to confirm the existence of the triad. The use of the term triad derives from the strong inter-relationships among its components.[3] For example, inadequate caloric intake relative to exercise energy expenditure can lead to suppressed reproductive function, but it may not be associated with low bone density if an individual has not been amenorrhoeic for sufficient duration, or if he or she possesses a genetic predisposition for initially high density.[3] Energy deficiency can also lead to reduced bone formation through pathways that are independent of effects on the ovarian production of oestrogen.[12 13]

DiPietro and Stachenfeld play down the importance of the triad and its specificity to athletes through repeated references to a recent paper by Torstveit and Sundgot-Borgen[14 15] that reported an incidence rate of the female athlete triad in Norwegian athletes that was similar to that observed in their age-matched controls. Although explanations of the methodological errors of this study have been published elsewhere,[16 17] a reiteration of these errors is warranted by DiPietro and Stachenfeld's failure to acknowledge them. The methods of Torstveit and Sundgot-Borgen lead to their underestimation of the incidence of the triad in athletes and the overestimation of its incidence in controls. For example, Torstveit and Sundgot-Borgen[14 15] underestimated energy deficiency by defining disordered